Letters to the Editor

Malakoplakia and endocrine abnormalities

Sir,

I was interested to read the paper by McClure *et al.* on the association between malakoplakia and hypercortico-steroidism.¹ I also have had experience of malakoplakia in an elderly patient in whom there was morphological evidence of endocrine abnormalities.

At post mortem examination on an 84 year old female, it was noted that there was severe facial hirsutism, bilateral temporal recession of head hair and breast atrophy. One ovary was absent, the other showing post-menopausal atrophy and there were no obvious tumours in the genital system. There was a 3 cm diameter cortical adenoma of the left adrenal. The left kidney showed pyelonephritis, stones and a purulent exudate, culture of which grew *Escherichia coli*. Malakoplakia was seen as distinct plaques within the bladder.

In this case, the appearance of virilisation could well be explained by diminished ovarian function. There was no evidence that the virilisation was due to the adrenal adenoma, although such an association is well-known.² While adrenal cortical adenomas are not infrequent findings during necropsies, adenomas larger than 2.5 cm diameter are distinctly uncommon.³ The association between malakoplakia and an adrenal adenoma may be entirely fortuitous but, in the light of the observations of McClure *et al.*¹ linking malakoplakia and endocrine abnormalities, a significant relationship is a possibility.

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Ruptured ascites: 'accidental' massive paracentesis without complication

Sir,

We report a case which supports the recent views^{1,2} that paracentesis is a safe option in managing massive cirrhotic ascites. A 52 year old man, with longstanding massive tense ascites, was sent to hospital having lost 'two bucketfuls' of

fluid through a spontaneous rupture in a pre-existing umbilical hernia. A weight loss of 14.7 kg from a clinic visit 3 days previously was recorded. On admission he was in no distress and vital signs were stable. The most striking feature was a non-tender, reducible umbilical hernia, 8 cm wide, with a 4 cm linear rent at its apex, leaking clear fluid. Gross peripheral oedema was noted but there were no signs of cardiac failure. Plasma creatinine, blood urea and electrolytes were normal and septic screen of the ascitic fluid was clear. Serum albumin concentration was 23 g/l. Biochemical liver function tests remained unchanged from 3 months previously.

Fluid leakage was collected in ileostomy bags and intravenous cephadrine and metronidazole given as prophylaxis against bacterial peritonitis. Bed rest and salt restriction were enforced, and additional vitamin K administered. The peripheral oedema cleared within 72 hours of admission. At no time was oliguria noted, and renal function remained stable throughout the 2 week hospital stay. The ascites was further controlled with frusemide and spironolactone in moderate dosage.

A recent study² recorded no instance of hypotension of azotaemia following 5-litre paracentesis in eighteen cirrhotics with tense ascites and peripheral oedema. It appears that mobilization of peripheral oedema fluid³ in such cases counteracts any hypovolaemia due to paracentesis. This was probably the case in our patient.

The Spanish experience^{1,2} appears to disprove the contention that therapeutic paracentesis carried great risk of hypovolaemia, renal failure, dilutional hyponatraemia and encephalopathy. The other lesson from this case is that controlled paracentesis may prevent the life-threatening complication of hernia rupture in ascites, where diuretics and salt restriction are ineffective.

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